UNCOMMON PRESENTATION OF A LONG-STANDING CALCIFYING FIBROBLASTIC GRANULOMA IN THE POSTERIOR MANDIBLE

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Abstract

A 14-year-old Malay female was referred in view of a five-year history of an enlarging swelling over her lower right teeth, causing a disturbance in eating, speaking, and reciting Quranic verses. Clinical examinations revealed a non-tender, pedunculated exophytic growth on the buccal site measuring approximately 1.3 cm x 1.5 cm and the lingual site approximately 2.9 cm x 2.5 cm in size. The lesion encompassed her right mandibular first molar and second premolars with pinkish-red overlying mucosa and ulcerations on the lingual and occlusal aspects of the swelling. The right mandibular first molar had grade I mobility and 5 mm periodontal pocket depth associated with grade I furcation involvement. Deep scaling was performed on the involved area, and the excisional biopsy was performed under general anaesthesia. Histopathological examination revealed cellular connective tissue with focal calcifications, which gave a final diagnosis of Calcifying Fibroblastic Granuloma (CFG). The patient had an evaluation for oral hygiene maintenance and phase I periodontal treatment, which included deep scaling and a prescription for chlorhexidine mouthwash. No recurrence was observed one year post-operatively.

Keywords: Calcifying Fibroblastic Granuloma, Gingiva, Surgical, Focal Calcification

Introduction

A swelling or lesion in a child's mouth can be very concerning for both the child and their parents. It causes further concern when the lesion appears to be increasing in size. Calcifying fibroblastic granuloma (CFG) is one such lesion that can manifest as a swelling in the gingiva (1). CFG is the third most prevalent lesion among all localized reactive hyperplastic lesions, following pyogenic granuloma and giant cell central granuloma, accounting for 2-9% of all gingival lesions (1).

Clinically, CFG arises as a solitary, focal exophytic mass exclusively on the gingiva, commonly appearing to originate from the interdental gingiva (1, 2). The growth can be sessile or pedunculated, smooth-surfaced, usually firm, and non-tender to palpation in most cases. Histopathological examination revealed features of parakeratinized stratified squamous epithelium, fibrocellular of underlying connective tissue, and bony trabeculae formed in the central portion (3).

The treatment of choice for CFG is total excision with peripheral and deep margins, including the periodontal ligaments and the affected periosteum at the base of the lesion, to reduce the chance of recurrence (2, 3). Here, we present a case report of a 14-year-old female adolescent with a large CFG in her right mandible. Due to its rare incidence in the posterior mandibular region, the description of the long-standing swelling by the primary healthcare clinic and who treated this case is relevant and important.

Case presentation

A healthy 14-year-old female adolescent came with a chief complaint of two lumps on her lower right gingiva for the past five years. The swellings were painless but caused

discomfort, especially upon eating, speaking, or reciting Quranic verses. The swelling was primarily nodular, which gradually increased in size and attained its present size. The patient denied any history of trauma, injury, or food impaction in situ.

Extraoral examinations revealed a symmetrical face with a tender, palpable, and mobile right submandibular lymph node measuring 0.4 cm x 0.2 cm. Intraoral examinations revealed a solitary growth noted on the buccal and lingual aspect of the gingiva in relation to the lower right first and second premolars (teeth 44 and 45). The lesion on the buccal site was approximately 1.3 cm x 1.5 cm in size, pedunculated, and rubbery in consistency. The lesion on the buccal gingiva appears pinkish and is located at the interdental papilla of teeth 45 and 46 (Figure 1A). In addition, there is an exophytic solitary nodule on the lingual site of teeth 44 and 45. There is a lesion measuring approximately 2.9 cm x 2.5 cm, ulcerations on the upper surface, non-tender on palpation, and firm in consistency (Figure 1B). Localized supragingival and subgingival calculus were present on the lingual segment of the affected teeth. Periodontal Pocket Depth (PPD) measuring 5 mm was noted on the mesial aspect of tooth 46 and 4 mm pocketing on the distal site of tooth 45 with mobility grade I on both teeth.

An intraoral periapical radiograph showed interdental infrabony bone loss mesial to tooth 46 approximately 5 mm, mesial and distal widening of the lamina dura of 45, and grade I furcation involvement on 46 (Figure 2A). Clinically, the lesion's differential diagnoses were include pyogenic granuloma, peripheral odontogenic fibroma, fibroma, and peripheral giant cell granuloma. Deep scaling was performed on the affected area to eliminate the local irritants, and an excisional biopsy was carried out using general anaesthesia. Intraoperatively, the lesion appeared to arise from the interdental papilla of teeth 45 and 46, extending to buccal and lingual mucosa (Figure 3). Post-operatively, the patient has been prescribed a tab of Paracetamol 1gram qid/prn for five days, mouthwash chlorhexidine, and gengigel. Oral health care was emphasized by involving twice-daily tooth brushing, the use of dental floss, and chlorhexidine mouthwash. The patient was advised to use the single-ended tufted toothbrush to clean the site post-operation to ensure effective cleaning. Deep scaling was performed 3 months after the operation. Histopathological Examination (HPE) of the lesion showed hyperplastic parakeratinised stratified squamous epithelium exhibiting extensive ulceration covered by the fibrinopurulent membrane, engorged areas of blood vessels, and evidence of ossification in the connective tissue. The definitive diagnosis of calcifying fibroblastic granuloma was made based on clinical and HPE findings.

At 12 months review, the PPD on the mesial side of 46 was reduced to 2 mm with no mobility of teeth 45 and 46, no furcation involvement of 46, and no recurrence of the growth (Figure 4). However, the gingival recession still persists. Periapical radiographs show evidence of bone formation at the bony defects on both teeth 45 and 46 (Figure 2B).

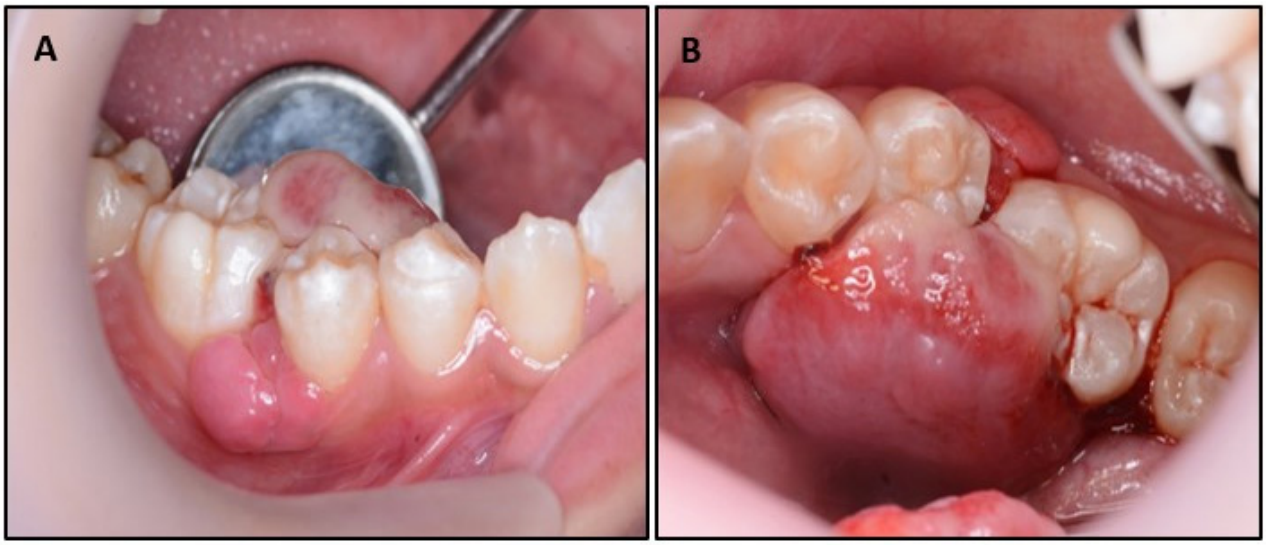

Figure 1: Solitary exophytic mass on the posterior mandibular gingiva **(A)** Buccal view; **(B)** Lingual view

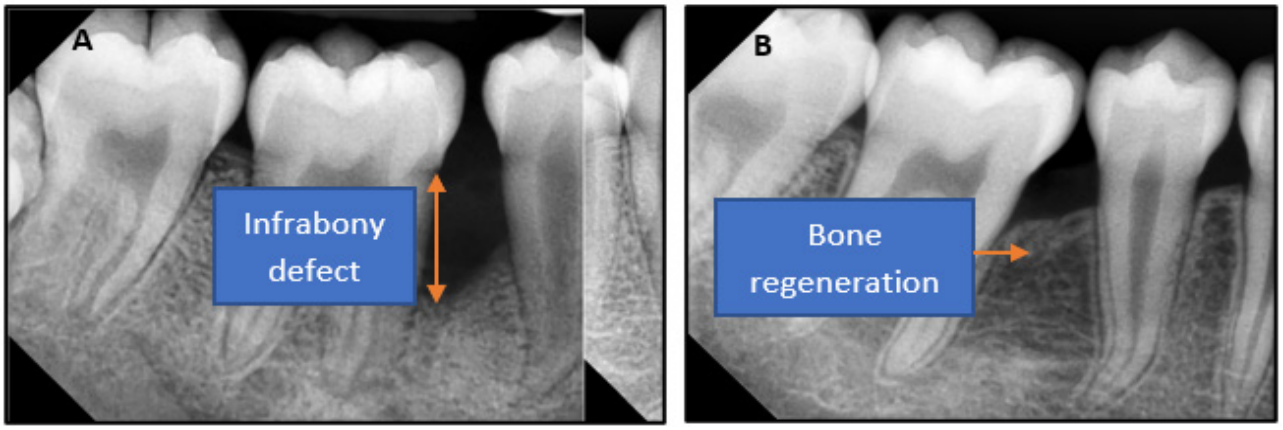

Figure 2: (A) Pre-operative periapical view of mandibular right first molar shows 5 mm of intrabony defect; **(B)** Post-operative after 12 months, bone regeneration and apparent reformation of periodontal ligament in the apical part of the infrabony defect on the 46

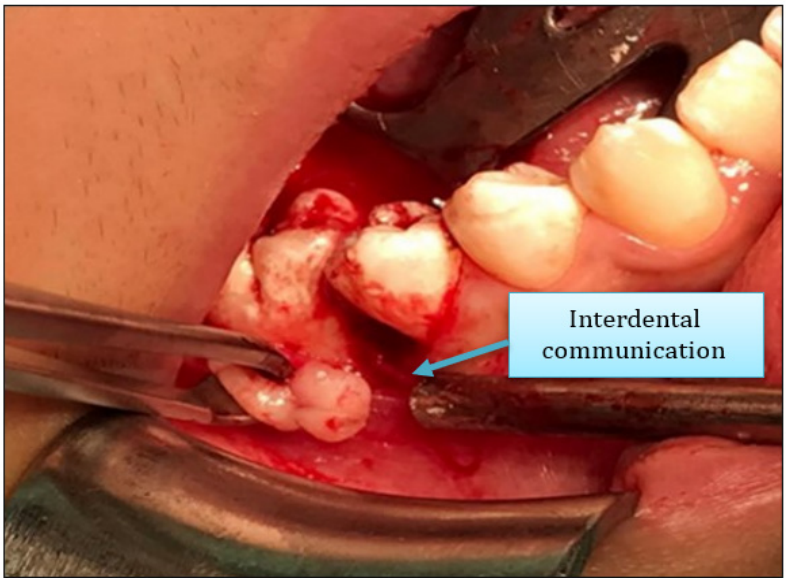

Figure 3: Interdental communication between buccal and lingual lesions

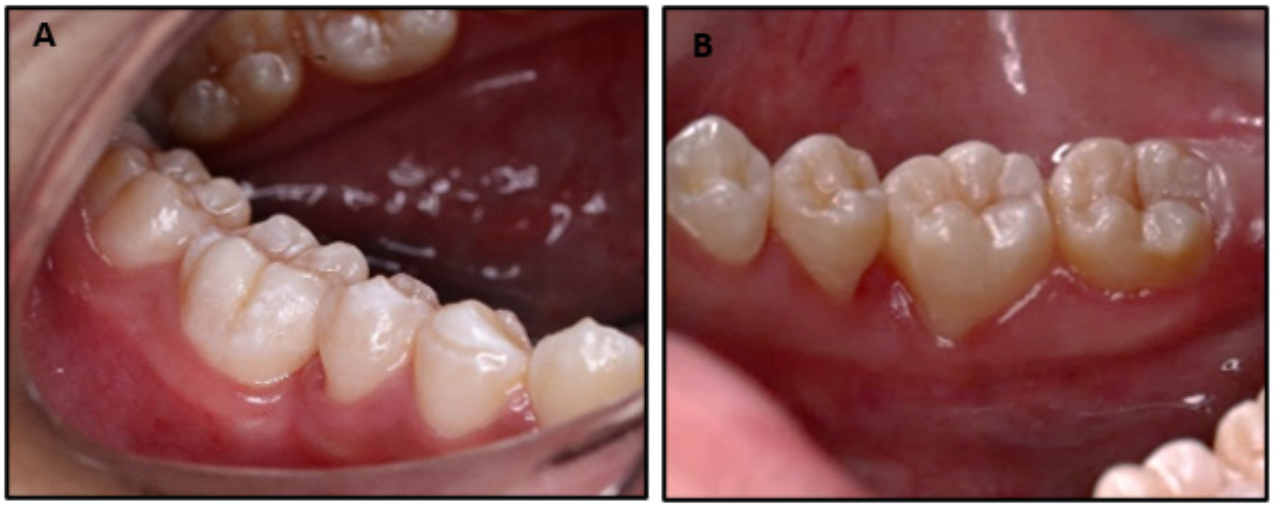

Figure 4: Postoperative view 12 months on the **(A)** Buccal site; **(B)** Lingual site

Discussion

CFG is a reactive inflammatory hyperplasia exclusively to gingival tissues (3). The etiology and pathogenesis of CFG are unknown (4). Researchers hypothesize that some CFG begins initially as pyogenic granulomas that undergo fibrous maturation and eventual calcification (4). Additionally, CFG seems related to periodontal ligaments, as it exclusively develops in the gingiva (interdental papilla) and because of its proximity between both tissues (2). Additionally, the microfibrils in periodontal ligaments, known as oxytalan fibers, are detected within the mineralized matrix of lesions (4).

The aetiopathogenesis of CFG has been linked to trauma and local irritants, including deposits of calculus and plaque and ill-fitting dental appliances that are likely connected to trauma or local irritants (3, 4). This is in accordance with the presented case, as the patient manifested with supragingival and subgingival calculus on the site of the lesions. Furthermore, some authors correlate the appearance of CFG with hormonal changes as it has a higher female predilection, frequent occurrence in the first decade of life, and decreased incidence after 30 years of age (5). This might be so in the present case, as the patient was female and diagnosed at 14 years of life. Up to two-thirds of these lesions are commonly seen in the maxilla than the mandible and favor the interdental papilla of the incisors or canine region (1, 4). In this case, the lesion in question may be deemed uncommon in terms of location because it affects the posterior mandible.

Regarding the extension of the lesion, in most cases, the longest length does not exceed 1.5 cm. However, some authors have occasionally described the unusually large size of CFG lesions from 6 to 9 cm in diameter (2, 3). There have been reports of tooth migration, separation, and bone deterioration in bigger lesions (4). These uncommon clinical characteristics were seen in this case report, where massive growth of the lesion has caused the destruction of the supporting tissues of the involved teeth and caused tooth displacement 45.

Clinical diagnosis can be difficult because there are several other lesions whose clinical signs can resemble CFG (4). To illustrate, during the second decade of life, CFG and pyogenic granuloma are often seen as slow-growing, solitary lesions on the attached gingiva, usually in the upper anterior region. The lesions may appear reddish to pink in color, less than 2.5 cm in diameter, and sessile or pedunculated (5). Compared to CFG, pyrogenic granuloma rarely exhibits calcifications, tooth displacement, or resorption of alveolar bone (4, 5). Additionally, peripheral giant cell granuloma (PGCG) has similar clinical features to CFG. However, PGCG has a purple-blue hue on its surface and most commonly occurs during the 4th to 6th decade of life (4).

On radiographs, CFG may cause thickening of the lamina dura and widening of the periodontal ligament gap around the affected teeth. A significant proportion of patients show no obvious superficial bone erosion (4). Aggressive bone loss is visible in the current case because of the long-standing presence of the CFG (3).

The confirmatory diagnosis of the lesion is made by histopathological examination (4, 5). Histopathological examination of the specimen revealed parakeratinized stratified squamous epithelium, fibrocellular underlying connective tissue stroma, and bony trabeculae formed in the central portion (6).

The treatment for CFG is excision and complete removal of the lesion down to the bone, including adjacent periosteum and periodontal ligaments, to avoid recurrence (7). Dental plaque and calculus are considered a main aetiological factor capable of predisposing to the development of CFG (7, 8). Therefore, the patient presented in this case report has been placed under regular review for oral hygiene maintenance and phase I periodontal therapy. Interestingly, there was evidence of regeneration of bone and pocket depth reduction after a one-year follow-up. Scaling and root planing treatments combined with personal plaque control were found to be a beneficial therapy method in controlling the infrabony defect in this patient (7, 9).

Numerous injuries, insufficient initial clearance, or the persistence of local irritants have all been linked to the recurrence of CFG, with rates ranging from 8 to 20% or as high as 30.4%, according to published research (9, 10). Since the likelihood of lesion recurrence is high, the current patient was reviewed periodically for more than a year after surgery. It is critical to emphasize that rigorous postoperative follow-up is required for early diagnosis of CFG relapses.

Conclusion

The adolescent and her parents overlooked this long-standing lesion until she developed more severe discomfort during ordinary chores such as speaking, eating, and reciting Quranic verses. Thus, early diagnosis of CFG to prevent complications, along with surgical excision and curettage of surrounding tissue, is important for the prevention of recurrences. Daily oral hygiene maintenance coupled with frequent recall visits by patients is vital for the long-term success of periodontal phase I therapy. Close postoperative follow-up is mandatory due to the high recurrence rates.

Acknowledgment

We would like to thank the Director General of Health Malaysia for his permission to publish this article.

Ethical Clearance

The patient and her mother provided informed consent.

Competing interests

The authors declare that they have no competing interests.

Financial support

No funding was received for this work.

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